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THREE CASES OF PHTHISIS WITH CONTRACTED LUNG,

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Read October 23rd, 1868.

CASE I.

WILLIAM DOWNER, æt. 40. Admitted into the Brompton Hospital, under the care of Dr. Powell, July 18, 1868, during Dr. Sanderson's absence from town.

History.—Patient aged 40, married. A carpenter by trade; both parents living, healthy; one brother died of phthisis. Patient was quite well till six years ago, when he had a cough; this ceased, and he then remained well until twelve months ago, when he had inflammation of the lungs, from which he has never recovered. In February last (six months ago) he had hæmoptysis to the amount of two pints, and he has occasionally spit blood since; has been losing flesh for twelve months. Patient now coughs, with abundant mucopurulent expectoration, nummulated and floating in a clear fluid. Appetite fair, bowels costive. He is slightly emaciated, but has not at all the aspect of phthisis; complexion pale with a straw tint.

Physical signs.—No movement with inspiration of left side of chest. Percussion (left side) dull anteriorly from apex to

fifth rib, and to within an inch and a half of the middle line, where the resonance of the opposite lung commences. Respiration harsh, feeble in subclavicular region. At the outer part of the third rib, distant, tubular, with cavernous clicks on cough, and bronchophony. Feeble below.

Heart's apex beats in the fourth interspace, two inches to the left of the nipple line; sounds normal. Stomach

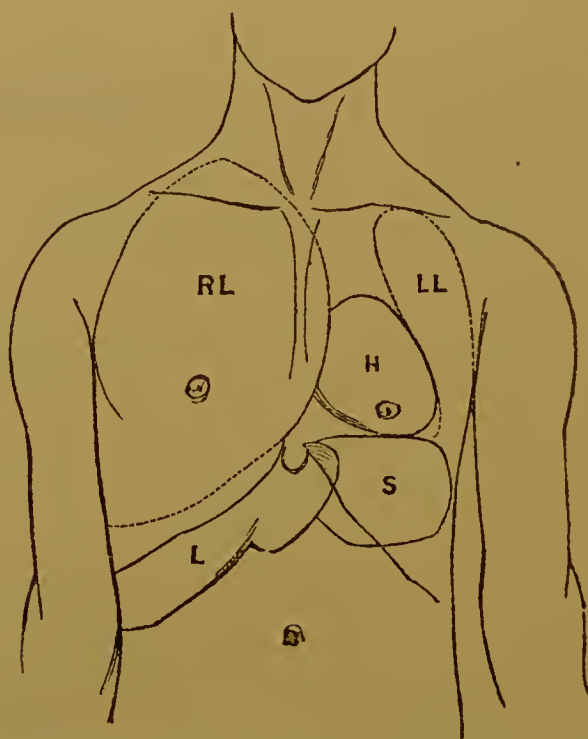


FIG. 1 (illustrating Case I.)—R.L. Right lung, reaching across median line. L.L. Left lung contracted, leaving pericardium uncovered. H. Heart, apex at 4th interspace to left of nipple. S. Stomach drawn up to 5th rib. L. Liver.

resonance obtained below the fifth rib. Posteriorly—in supra-spinous fossa dullness, with bronchial respiration and bronchophony. Scapular region—dull, with tubular respiration and cavernous cough. Base, more resonant to the axillary line, respiration feeble, harsh, but with more vesicular quality than elsewhere.

At the right apex the respiration is harsh and divided with prolonged expiration.

Liver dullness unchanged, no enlargement of spleen detected.

Diagnosis.—(See Fig. 1 showing position of organs.) Left

lung contracted, indurated, with a long vertical cavity occupying the posterior part of the lung. Pleura greatly thickened. Right lung enlarged by compensatory hypertrophy, but becoming involved at the apex and anterior margin.*

Progress.—Patient, soon after admission, July 24, had an attack of copious hæmoptysis (one pint), which was repeated on July 27, and prevented a thorough examination of the chest

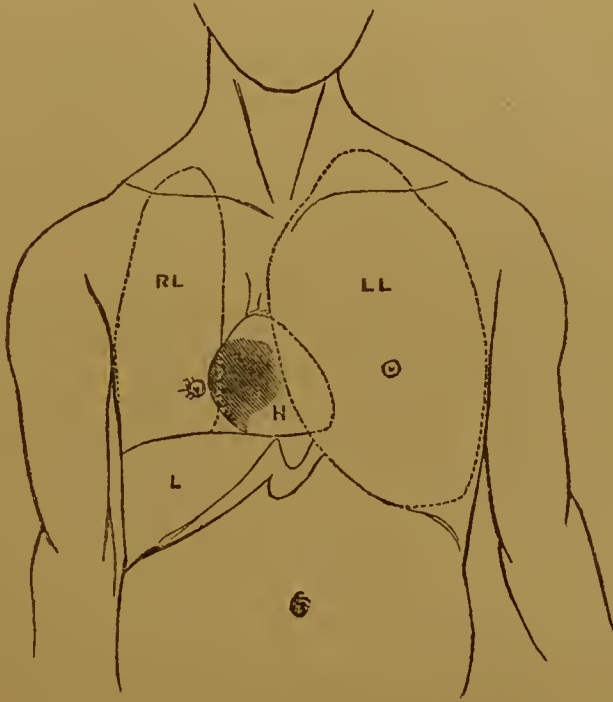


FIG. 2.—R.L. Right lung contracted, leaving pericardium uncovered. L.L. Left lung, extending across median line. H. Heart, area of impulse and dullness indicated by shading. L. Liver.

until July 31, when the above notes were taken. The hæmoptysis was repeated on August 6 and 8, the sputa having been more or less tinged in the intervals, and the patient died with a fifth attack of hæmoptysis on August 9.

Treatment.—Astringents.

Post-mortem examination twenty-four hours after death; weather very hot; body not emaciated; a considerable layer of adipose tissue covering the muscles. On removing sternum and cartilages, the right lung is seen to extend across the median line, its anterior margin opposite the third cartilage,

* An exact diagnosis as to the nature of the disease was not arrived at during life.

reaching a line drawn vertically downwards from a point $\frac{3}{4}$ in. to the left of the sterno-clavicular articulation.

The left lung opposite the second rib reaches by a thin hard margin to within $\frac{3}{4}$ in. of the line of sterno-clavicular articulation, then slants backwards to the axillary line, becoming invisible from the front. The heart's apex is opposite the interval between the fourth and fifth ribs in the anterior axillary line. Inferiorly the diaphragm arches upwards to the level of the fifth rib.

Heart healthy, rather large, the right cavities somewhat increased in size. Pulmonary artery measures across valves 3 inches; aorta ditto.

Left Lung.—Very firmly adherent, removed with great difficulty.

Anterior margin of lung is thin, tough, leathery feeling, covered by a considerable quantity of fat between the pleura and pericardium; this fat is more abundant above, and contains some deeply pigmented glands. On section this portion of the lung is of an iron-gray colour, with ramified points of pigmentation; on closer inspection numerous very minute round glistening granules are seen looking very like small gray granulations. The margin of the lower half of the lung is still firmer and excavated.

Lateral part of upper lobe. The thickness of the pleura varies from $\frac{1}{4}$ to $\frac{1}{2}$ an inch; immediately under the pleura is a thin layer of hardened, very tough, gray pigmented lung tissue, at parts thinned down by excavation to the pleura, at others thicker and prolonged inwards, forming horizontal septa which divide one cavity from another. Opposite the septa the pleura is also thicker and prolonged a little inwards. Scattered through the deeply pigmented tissue, and also in the pleura, are seen similar glistening points to those mentioned above.

The lateral part of the lower lobe is tough, collapsed, and sinks in water, but is more spongy in texture than the corresponding part above. The pleura covering it is from $\frac{1}{16}$ th to $\frac{1}{8}$ th of an inch in thickness, and numerous fine processes are seen directed downwards from the interlobular septum, and less plainly inwards from the parietal pleura. The tissue is seen to contain many granulations, a large number of which are situated on the fibrous processes.

Posterior part, pleura greatly thickened at the apex, $\frac{3}{4}$ in. at its thickest part. Three principal excavations occupy

from above downwards this part of the lung. They are bounded by a thin layer of pigmented consolidated lung tissue, sending septa inwards, which separate them from one another. The interlobular septum, with a layer of lung tissue on each side of it, forms one of these partitions. The cavities contain but very little secretion, some clotted blood, and are lined by a smooth and tolerably tough false membrane. These cavities communicate widely with bronchial tubes. In the lowest and smallest cavity is seen at its anterior part a small pulmonary aneurism, size of a pea, the vessel becoming obliterated at a distance of $\frac{1}{2}$ an inch beyond the dilatation. At quite the posterior part of the lower lobe, and at a small portion below the lower cavity, the pulmonary tissue is more spongy than elsewhere, but collapsed.

The right lung—perfectly free from adhesions. The pleura healthy, with the exception of one or two gray granulations seen on its surface. The lung is much enlarged and emphysematous. On section at the apex are seen, pretty numerous scattered, gray granulations,* hard, shotty feeling, surrounded by emphysematous tissue. The posterior part of the apex and also of lower lobe congested. At one spot in the apex is a dry, almost cretaceous encysted mass about the size of a pea. There are also gray granulations scattered through the upper lobe and very thinly through the lower.

Liver	}	not obviously diseased — not examined microscopically.
Kidneys		
Spleen		

Intestines—not examined.

Microscopical characters.—Less diseased part from base of lung. A section taken from this portion shows a general groundwork to consist of cells and nuclei, varying in size from the large connective tissue cell to the tubercular nucleus or cell; these cells and nuclei are mixed up together without any very definite order, but in the walls and neighbourhood of bronchial tubes, vessels, and collapsed alveoli, the nuclei are more densely crowded. There is a delicate, ill-defined, and faintly granular fibrous stroma, in which the

* It is worthy of remark that many of these granulations are slightly larger than the miliary granulations of acute general tuberculosis, many of them also have a more angular form; they are identical with the typical gray granulations in every other respect. This tendency to angularity I have several times observed in cases marked by chronicity.

larger cells give off one or more fine processes, at favourable portions of section being seen to form by the communication of these processes complete networks. Here and there are portions of lung tissue, where the alveoli are patent and perfectly healthy, but they are for the most part collapsed, and their walls thickened, forming stellate fibro-nuclear masses. The alveoli are at this part remarkably free from large granular cells (epithelial pneumonia), all those affected being, as above said, simply collapsed and their walls thickened.

In the dense tissue surrounding the cavities, the fibrous stroma is strongly marked, the fibres appearing broad, flattened, homogeneous, intercommunicating, and in the interspaces, and on the fibres themselves, are innumerable nuclei or cells similar in size and appearance to tubercular nuclei. These nuclei are at parts densely crowded together, excluding the fibres, or only admitting fine processes between them. In favourable situations, the large connective tissue cells, with their communicating processes, can be readily distinguished. There are also connections of pigment scattered over the field. Many of the alveoli here are seen to contain large granular pneumonic cells, their walls being much thickened by the fibro-nuclear growth.

Comments.—The history of this case points to the disease being tubercular and having an inflammatory origin, and the interpretation of the morbid appearances that I would suggest is the following:—

There has been old arrested disease, which has produced the excavations now dried up; succeeding to this there has been a second disease, differing from the first not in kind, but in rapidity of progress, this difference arising from there having been in the first attack some intra-lobular complication (catarrhal pneumonia). This second disease is indeed a true interstitial pneumonia accompanying the tubercular granulations. Finally, in the opposite lung, the disease reassumes its common character of gray granulations without any fibrous change associated with it.

The case is remarkable for the extent to which the interstitial pneumonia has proceeded, less degrees of this pneumonia being common in all cases of chronic tubercular phthisis. The distinction clinically between this case and one of cirrhosis is obvious, there being excavation of two-thirds of the lung, a careful physical examination being alone necessary.

Chronic pneumonia and pleurisy were excluded by the disease being most advanced at the upper part of the lung.

The distinction during life between this disease and that described by Dr. Clark as Fibroid Phthisis, would be very difficult, for though there was evidently disease at the apex, it was less marked here than at the third rib and scapular region. Moreover though, at the time this case was examined, the other apex was evidently involved, yet the presence of this sign is a mere question of time, and it might not have been present a month earlier while the disease was in full progress. There was no cardiac murmur in this case; the presence or absence of cardiac murmur in these cases has never, I think, been satisfactorily explained, for it is not uncommon to meet with cases of very considerable displacement of heart without any murmur, and a murmur may be present at one time and absent at another.

This patient died directly from hæmoptysis, while his lung disease was yet in mid career. There was therefore no opportunity for any treatment, and I would therefore reserve the few remarks I have to make on that subject for another communication.

The cause of this hæmoptysis was found to be the rupture of a small dilatation of a branch of the pulmonary artery. This mode of occurrence of hæmoptysis has attracted much attention of late, and is now recognised as one of the most common causes of fatal hæmoptysis, as it certainly is also an occasional cause of hæmoptysis which is not fatal. Of eight cases of fatal hæmoptysis which have been carefully examined post-mortem at Brompton since February 1868, in six an aneurism of a pulmonary vessel has been the source of hæmorrhage.

CASE II.

Thomas Joiner, æt. 38, admitted an out-patient under my care, at the Brompton Hospital, May 30, 1868.

Family history.—Grand-parents lived to a great age. Father died aged 84, and mother aged 76, of natural decay. No uncles or aunts have suffered with any chest affection. Patient has four sisters and one brother living, has lost two brothers; one, aged 50, died of dropsy, the other, aged 31, of a tumour on the back.

Personal history.—A gardener, of regular habits, has been

married sixteen years, no family, wife has had no miscarriages. Patient had slight gonorrhœa many years ago, never syphilis, never suffered from fistula. He was laid up five years ago with some head affection; some pills were given him which caused him to sweat profusely; he got up and went downstairs afterwards in his shirt while damp with perspiration, and got a chill, which increased the severity of the head symptoms; he states that he did not suffer from the chest at this time; was laid up for seven weeks. In January, 1868, he suffered from chilliness and sweats, with pain across the chest of a darting character and slight cough; he kept upstairs for fourteen days, and was confined to bed for one week; has been ailing since. Expectored a few lumps of blood in April; has been getting thinner. Complains now of occasional cough, slight expectoration, pain in left lower axillary region. Appetite good, pulse moderate, tongue thinly coated, bowels regular.

Patient is a medium sized, rather spare man, with an oval face, clear complexion and fresh colour, hair abundant, dark.

His chest was first examined June 13, but the physical signs then noticed only differed from those stated in the present note in a few particulars to be afterwards mentioned.

Oct. 7.—Has improved considerably in appearance, has gained some flesh.

Pulse 100.

Marked lowering of shoulders. L. nipple $\frac{7}{8}$ in. lower than R.

R side measures, 1 in. below nipple, 17 in.; expansion $\frac{3}{4}$ in.

L " " " 16 " " nil.

R " 1 in. above " 19 " " $\frac{1}{2}$ in.

L " " " 17 " " $\frac{1}{4}$ in.

Mid sternum to R nipple $4\frac{1}{2}$ in., to left nipple $3\frac{7}{8}$ in.

Left side.—No expansion, decided flattening infra-clavicular region and infra-mammary region, slight bulging axillary region. Heart's impulse extended, apex beats a little outside nipple in fifth interspace.

Percussion, in nipple line, wooden to fourth rib, dull from fourth to lower border of fifth, where stomach resonance is obtained. Axillary region, dull posterior to a line extending obliquely downwards and forwards from mid axilla to below nipple. Supra-spinous fossa hyper-resonant to spine of scapula and in interscapular region to within two inches of angle

of scapula, dull elsewhere, except within an inch of spinal column, where there is some resonance.

Respiration, supra-clavicular region, harsh, wavy, with prolonged expiration. Infra-clavicular region imperfectly bronchial and jerking with bronchophony; at the second and third ribs, nipple line, very bronchial and jerking, very feeble elsewhere anteriorly. In the axilla respiration is bronchial and blowing above, feeble with bronchial expiration below. Supra-spinous fossa bronchial with conducted whisper, more intensely bronchial just below spine of scapula with pectoriloquy. Feeble and bronchial below, a few clicks were heard after cough near angle of scapular. Near the spinal column, respiration is vesicular and exaggerated.

Vocal fremitus lessened both anteriorly and posteriorly, more particularly at the lower part.

Right side.—Hyper-resonant, the resonance extends beyond median line above, reaching $\frac{3}{4}$ in. to left of sterno-clavicular articulation, and slants downwards, so that at fifth cartilage it is $\frac{1}{2}$ in. to left of middle line. Liver dullness commences at seventh rib.

Respiration below and above the right clavicle, and also in the supra-spinous fossa, harsh, divided, expiration and inspiration equal, elsewhere exaggerated vesicular.

The differences between the physical signs noted now and those of June 13, are—

Left side.—At the apex percussion now more resisting, and respiration more bronchial. At the third rib there were large clicks to be heard; these have now disappeared.

Right side.—At the apex, where before simply exaggerated, the respiration is now harsh, divided with prolonged expiration.

Oct. 21.—On again examining the right apex, the respiratory sounds were as before; there was some moist crackling to a slight amount heard by Dr. Sanderson and myself, above the clavicle.

23.—Urine thick, cloudy, faintly acid, sp. gr. 1028, clears on boiling, no albumen. On adding nitric acid after boiling effervesces freely and becomes of purplish tint. Another portion clears on adding liquor potassæ, and its colour remains unchanged, or but slightly deepened on boiling with the alkali.

Remarks.—Whether the patient had any chest complication with his illness five years ago, it is impossible to say;

the medical gentleman who attended him has, unfortunately, no notes of the case. The patient dates his present illness from an acute attack he had in February. This attack was probably one of pneumonia of a mild character, but which has never cleared off. This case is, probably, very similar in character to the preceding, being one of chronic tuberculation, with much interstitial pneumonia, supervening upon an acute inflammatory disease of the lung, affecting the base and very likely simple in its nature. The peculiar slow growth of the secondary disease, and the abundance of fibre growth associated with it, appear to be accounted for by (1) the constitutional inaptitude of the patient and (2) the mechanically quiescent condition of the part affected, being most favourable for uninterrupted slow growth.

The disease appears to be now invading the other lung in its usual form of gray granulations, unaccompanied with any excessive connective tissue growth, and at its usual seat, the apex of the lung.

27.—The temperature has been taken for five days since patient has been in the Middlesex Hospital, and it is remarkable that it has never been above 98° F., and the pulse and respiration have been quiet; although the physical signs seem unmistakably to point to slowly but steadily advancing disease in the opposite lung.

A short blowing systolic murmur, most audible a little above the apex near sternum, pointed out to-day by Dr. Clark.

CASE III.

William Clay, æt. 57, admitted into the Brompton Hospital, under the care of Dr. Sanderson, August 15, 1868.

History, &c.—Patient is a farm labourer, of temperate habits, married, has six children living, two died young, one aged about seven of some lingering illness, the nature of which is unknown to him. Grand-parents both lived to a great age. Mother now living, healthy, aged eighty-seven. Father died, aged sixty, of cold, having been ill only one week.

Patient has one sister living, healthy, has lost two brothers—one, a soldier, of black fever; the other a hard drinker, who died after two months' illness; he had slight cough, but no wasting; has lost two sisters, both of whom died in child-

bed. Patient was a strong and healthy man up to February last, having had no previous illness except occasional lumbago; never had the venereal disease. In February he caught cold from getting his feet wet; had some shivering, and pain in the left side, and was feverish, but did not keep his bed; had a slight cough, but no expectoration. This illness lasted six weeks, but he states that he never quite recovered it. He has never had hæmoptysis nor diarrhœa. Complained on admission of pain in the left side and shortness of breath, no cough nor expectoration, slight emaciation.

Appearance.—A stoutly built man of about middle stature. Complexion pale with a sallow tinge, scanty growth of hair, expression stolid without animation, no gouty deposits in ears.

Physical examination of the chest.—Left side, no expansion, shoulder lowered; supra and infra-clavicular regions, percussion resisting with deficient resonance to lower border of second rib, respiration very harsh, jerking, with prolonged expiration. From second to lower border of fifth rib absolute dullness. At the outer part of second interspace respiration very tubular with a few clicks and pectoriloquy; these sounds become less marked and more distant as examination proceeds downwards. The apex of the heart impinges at the fifth interspace three quarters of an inch to the left of nipple line. Heart sounds normal, stomach resonance is obtained below the fifth rib, nipple line. The resonance of the right lung extends to the left of middle line, reaching opposite the second and third cartilages the sterno-clavicular line.

On the right side very good resonance and expansion, with exaggerated breath sound. Liver dullness; on deep percussion sixth rib, superficial seventh, vertical dullness in nipple line three inches.

Posteriorly.—Left supra-spinous fossa percussion and auscultation give the same results as in the infra-clavicular region, except that the expiration is more prolonged and somewhat tubular. From the spine of the scapula downwards dullness absolute with no vocal fremitus. Respiration opposite the spine of scapula tubular, becoming more distant as examination proceeds downwards, altogether absent below the level of the eighth vertebral spine.

On the right side good resonance with exaggerated breath sound.

Average pulse 90 in the minute. Temperature taken for

four days in August 100° in the evening, 99° F. in the morning.

Re-examined, Oct. 3. — No appreciable difference in the physical signs on the left side. Flattening in infra-clavicular and supra-mammary regions well marked, with diminution in size of pectoralis muscle. To measurement the two sides are equal in circumference below nipple.

On the right side, where before the respiration was simply exaggerated, it is now, at the apex above and below the clavicle, harsh, divided; expiration equal in length to inspiration, and having some tubular quality. Elsewhere respiration exaggerated, with some harshness and dryness.

Oct. 4.—Pulse 88, respiration 20, temperature 99° F.

5.— „ 88 „ 20 „ 98·5° F.

13.— „ 96 „ 27 „ 98·8° F.

Urine sp. gr. 1020, pale, a very distinct cloud on boiling, not cleared by nitric acid, no casts detected; but on Oct. 14, by adding a little iodine to some sediment of the urine, and putting a drop under the microscope, many casts were seen, large granular, granules refracting, mostly with no epithelium, but here and there a single epithelium cell seen—some granule cells, pus cells, and a few blood cells seen. The casts were mostly of one size, but a few were very large and some small.

This case resembles, in many respects, one of fibroid phthisis of constitutional origin, viz., in the harshness and opacity of the skin, the very non-tubercular physiognomy of the patient, the clear evidence of renal disease, most probably of the contracted variety, and the signs of contraction and induration of one lung with thickening of the pleura; but the disease has commenced in the lung subsequently to an acute inflammatory attack;* it has invaded the whole of the lung, and I feel persuaded that the apex of the right lung is also involved from the great harshness of the respiration there; it would, therefore, seem to me to be of the same nature as Downer's case with the additional renal disease. It may, of course, be well argued that even should the opposite lung become more decidedly involved (as in Case II., Joiner), this may be a secondary or rather tertiary process of tuberculisation by inoculation from the cheesy deposits in the left lung. This explanation may be the true one, and it

* Dr. Spackman, of Farringdon, saw the patient when ill in February, and considered him to be suffering from pleuro-pneumonia.

might apply to all these cases of phthisis with contracted lung, rendering them all cases of engrafted tubercle; if true, however, we must consider the tubercle as engrafted upon the original inflammatory disease of the lung, not upon an intermediate fibroid affection, and in this view the chronic progress of the tuberculisation in the lung first affected, dependent upon its state of forced quiescence, with the later involvement of the opposite lung, would well account for the symptoms without any intermediate stage of simple fibroid disease.

General Remarks and Treatment.—The chief object in view in bringing these cases before the notice of the Clinical Society has been to elicit the opinion of the Fellows as to whether there is or is not a substantive disease to which the term ‘fibroid phthisis’ is applicable; a disease which is independent of tubercle,* or any definite local irritation, and which, when secondary to pneumonia or pleurisy, is in itself a progressive disease—i.e. a disease spreading beyond the limits of the primary lesion. I have placed the term ‘phthisis with contracted lung’ at the head of this paper, as indicating my own uncertainty on the question, and because I conceive it to be a more truly clinical term than ‘fibroid phthisis.’ The pure disease described by Dr. Clark under this term as of constitutional origin must be, I think, regarded as of very rare occurrence; while, on the other hand, the cases of ‘fibroid phthisis,’ considered by Dr. Clark as of local origin, are very common, and closely resemble one another in the physical signs which distinguish them; which physical signs are the result of a process of contraction of one lung, from whatever cause this contraction may arise.

All the cases of phthisis with contracted lung which have come under my own observation have had a local origin, the disease having supervened upon the cessation of some acute pulmonary affection, either simple basic pneumonia, or pleurisy, or tubercular pneumonia,† at one apex. Though limited at first to one lung, the other lung has later become affected in many, both basic and apex cases, and was so in

* By tubercle is meant a morbid growth, having its first stage in the gray granulation.

† By tubercular pneumonia is here meant tubercle, accompanied by much catarrhal or epithelial pneumonia; perhaps simple phthisical pneumonia should also be added.

all the five examples of which I have made examinations post mortem. The secondary disease has appeared to me to be chronic tuberculisations, in which the granulations have developed into a fibroid tissue (similar to that of the iron-gray induration of Addison), accompanied with more connective tissue growth (interstitial pneumonia) than is usually met with. The disease in the opposite lung has always been in the form of granulations, with more or less epithelial pneumonia, and some scattered lobular consolidations.

Several of the above cases are still under observation, and I hope, at some future time, to give some account of the experience derived from them.

The particular signs which are essential to all cases of phthisis with contracted lung, viz. flattening of the chest wall, displacement of organs towards the diseased side, and uncovering of the pericardium on that side, are all due to the shrinking of the lung, its vesicular tissue being gradually replaced by a solid fibrous material occupying less space. The great thickening of the pleura also, which gives the peculiarity to some of the physical signs, is the result of the same shrinking process: it cannot, I think, be regarded as truly inflammatory, but rather as a growth induced by the tendency to the separation of the pleural layers, and which is usually preceded by an earlier stage of serous effusion into a loose areolar tissue between these layers (œdema of the pleura). This stage of œdema can often be seen over a portion of lung less diseased, while the pleura above or below is converted into a solid thickness of dense fibrous tissue.* The position of maximum cardiac pulsation is liable, in some of these cases, to lead to error in diagnosis, since, when the right lung is contracted, the pericardium is uncovered on this side, and a systolic impulse is then felt and seen to the right of the sternum (see Fig. 2), and it may be very difficult or impossible to discover pulsation at any other spot, though the apex of the heart is really only a little more within the nipple than natural, but it is covered by the large left lung, or sheltered behind the sternum; hence the cardiac displacement appears much greater than it really is.

We learn from the study of these interesting diseases the extreme importance of making every effort to bring about complete recovery from acute inflammations of the lung or

* See 'Pathological Transactions,' vol. xx., case of chronic phthisis, illustrating one mode of thickening of the pleura.

pleura before the patient returns to the ordinary work of life; a change of climate for a time, in all doubtful cases, naturally suggests itself. Iodide of potassium, iron, and cod liver oil are well-known useful remedies. I think I have seen very great relief from the continued use of the iodine inunction, both in these diseases and in aiding recovery from ordinary chronic pneumonia and pleurisy; the ointment acts as a mild counter-irritant, relieving the dragging pains so commonly felt, and some of the iodine is, no doubt, absorbed, though in very minute quantity.* In some cases the cough is almost nil, in others very troublesome, and, as pointed out by Dr. Clark, often attended with vomiting; this is caused by the impaired structure of the lung and bronchial tubes, rendering expectoration very difficult; sedative inhalations are often useful. There are a few cases in which the cough is very paroxysmal, and comes on after meals, terminating in vomiting; in these cases strychnia is of great service.

* I was unable to detect any iodine in the urine of the patient Clay, who was treated in this way.

